

Using the Flea as a Weapon

By Mr. Reid Kirby

Deliberately spreading disease through arthropods is the vector effect of biological warfare. Its modern application in warfare started in the 1930s with Japan. Germany and the Soviet Union also conducted their own investigations in this area around the same time. During World War II, Canada pioneered the vector effect for the Allies.

Interest in vector weapons by the US Army Chemical Corps did not start in earnest until after the Korean War. Today, largely thought of as a throwback of the early days of biological warfare, the possibilities of the vector effect have emerged again after conjecture of the potential introduction of West Nile Virus to North America. Of the agent-vector combinations, the plague flea has the richest military heritage and is worth studying to understand this effect in biological warfare.

Natural History

Plague, a lethal epidemic disease since biblical times, has long been associated with rodents. The ancient Philistines made golden images of mice to ward off epidemics. It was not until the golden age of microbiology that plague was recognized as being caused by the microorganism *Yersinia pestis*. *Yersinia pestis* is named in honor of Alexandre Yersin, a student of Louis Pasteur, who isolated plague in patients in Hong Kong in 1894.

The role of the flea in spreading plague followed the discovery of the microorganism. Masanori Ogata of Japan first outlined the possible role of the flea in the spread of plague in 1898 (later confirmed by Paul-Louis Simond in France a year later). It was not until 1911 that medical entomology recognized the flea as the vector of plague.^{1,2} The rat was the most likely reservoir for the disease and easily defined the natural spread of the disease along trade routes.

Bubonic plague, the form transmitted by fleas, is so named due to the large oval buboes formed at the lymph node near the flea bite (such as in the groin or the armpit).

A rare secondary complication in natural epidemics is the occurrence of the lethal pneumonic plague (spread from human to human), which is an indicator of a biological attack when prominent. Pneumonic plague has a rate-of-action course of one to seven days, a duration-of-action course of one to two weeks, and a lethality rate of about 90 percent in one to two days.

The rate-of-action course of bubonic plague is two to eight days, with hospitalization required for up to ten days to avoid a relapse. In untreated cases, death usually occurs two to four days after the onset of symptoms. The bacteria rapidly spreads through the body, releasing endotoxins after reaching critical mass. The endotoxins cause the victims to go into shock and develop a high fever, rapid pulse, and low blood pressure. The mortality rate in untreated cases is 30 to 60 percent; treatment in state-of-the-art health care facilities reduces the rate to 5 to 15 percent. Though vaccines are available, they are generally only effective against moderate doses and, without semiannual boosters, provide only three months to a year of protection. Recovery from bubonic plague provides only a temporary immunity.

Plague epidemics occur naturally in endemic regions following an epizootic. Epizootics tend to occur in five-year intervals in endemic regions where fleas transmit the disease from rodent to human. Because of the role of the flea, epidemics tend to be self-limiting at temperatures under 45 degrees. The optimum temperature for fleas to transmit plague is 70 degrees; temperatures exceeding 85 degrees kill the bacteria. The temperature-dependent contagion explains why plague epidemics peak during warm, dry seasons and rapidly disappear with the onset of hot temperatures. Rainy seasons sharply reduce the incidences of plague.

There is a biomolecular basis for the temperature elasticity of epidemics. Several days after ingesting infected blood, fleas become blocked. Proliferated bacteria forms a clot that prevents food from entering the flea's stomach. Being famished, the flea attempts to feed through multiple

bites and regurgitates infected blood into its victim. An enzyme that contributes to the blocking of the stomach is active at 77 degrees. When the temperature rises above 98.6 degrees, the clot begins to dissolve. Blocked fleas can live up to 23 days in high humidity and normal room temperatures but die within three to four days from desiccation when exposed to optimum (moderate) temperatures.³

History has accounts of four plague pandemics: Egypt in 542, Asia Minor in the 14th century (which resulted in the death of nearly a quarter of Europe's population), Europe in the 15th and 18th centuries, and China in 1860 (an epidemic that continued to rage through the Vietnam War).⁴

Plague is an endemic disease in the western United States, with about ten cases a year reported by people who have had encounters with wild rodents. The first case occurred in San Francisco's Chinatown in 1900. The elimination of plague from endemic regions does not appear to be feasible, but reduced rodent populations, increased public education, and continued monitoring of flea counts on captured rodents will help with the fight.

Military History

The second plague pandemic draws the most interest from scholars of biological warfare. The plague epidemic in Asia Minor started from an epizootic in Mongolia and was carried westward by the Tartars. The spread to Europe may have resulted from a biological assault on the city of Kaffa. The Venetian historian, Gabriel de Mussis, described the siege of this trade center on the Crimean coast by the Tartars in 1346. After a three-year siege, plague broke out in the Tartar camp. Kaffa fell in 1348 after the Tartars catapulted their plagued dead over the city walls. The refugees that fled Kaffa by ship may have initiated the second plague pandemic that seeded throughout Europe from seaport to seaport.⁵

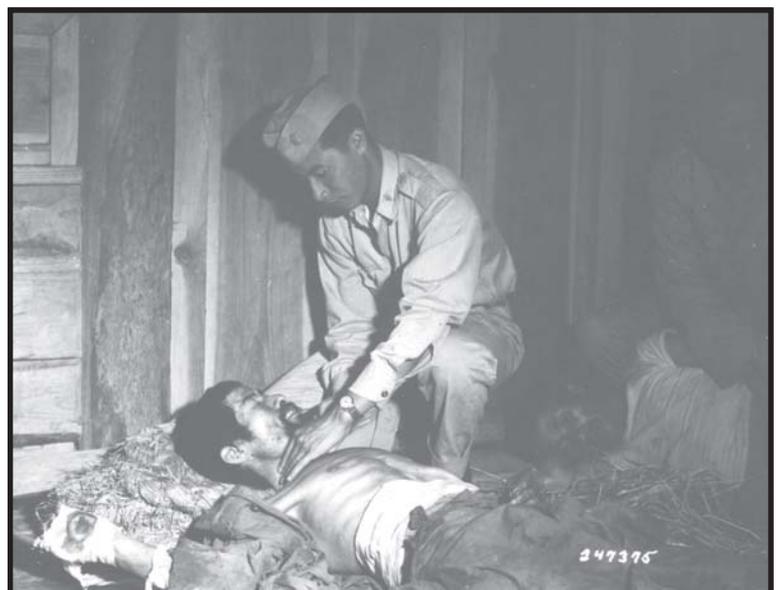
The impact of plague fleas during wartime (though unwittingly), continued in innumerable siege situations (like that of Kaffa) well after the Black Death period. In 1422, infected cadavers were catapulted into the city during the siege of Carolstein. Russian troops attempted to spread plague among Swedish forces using the bodies of plague victims.

It was not until World War II that plague fleas became a distinct biological weapon. Japan made the most pronounced efforts, followed by Canada and then the United States. It is probable

that other nations also used fleas as a weapon—as indicated in the infamous Hirsch report on German intelligence of Soviet biological warfare efforts—but there are too few details to recount.

The Japanese started their biological warfare efforts in the 1930s during their occupation of Manchuria and later during their invasion of China. At first, the Japanese experimented with sprays and bursting munitions to release bare germs for a lung effect. But the Japanese lacked an understanding of aerosols and respiratory pathology and soon changed their focus to disseminating plague using the human flea *Pulex irritans*. The flea protects plague bacteria from environmental strains and delivers a dose to its victims through bites. The Japanese experimented by employing plague fleas against China and contemplated use against the United States.⁶ The efforts to weaponize plague fleas were conducted under the auspices of Unit 731, the notorious Japanese biological warfare unit disguised as a water purification unit. In 1940, the cities of Chü Hsien and Ningopo were attacked with planes dropping rice, wheat grain, and paper packets of fleas. The drops resulted in an epidemic that killed 120 people. A similar attack followed a year later in Changteh City, killing 24 people. The Allies began investigating the activity of the Japanese, but the investigations failed to uncover any conclusive evidence. Suspicions continued throughout the war.

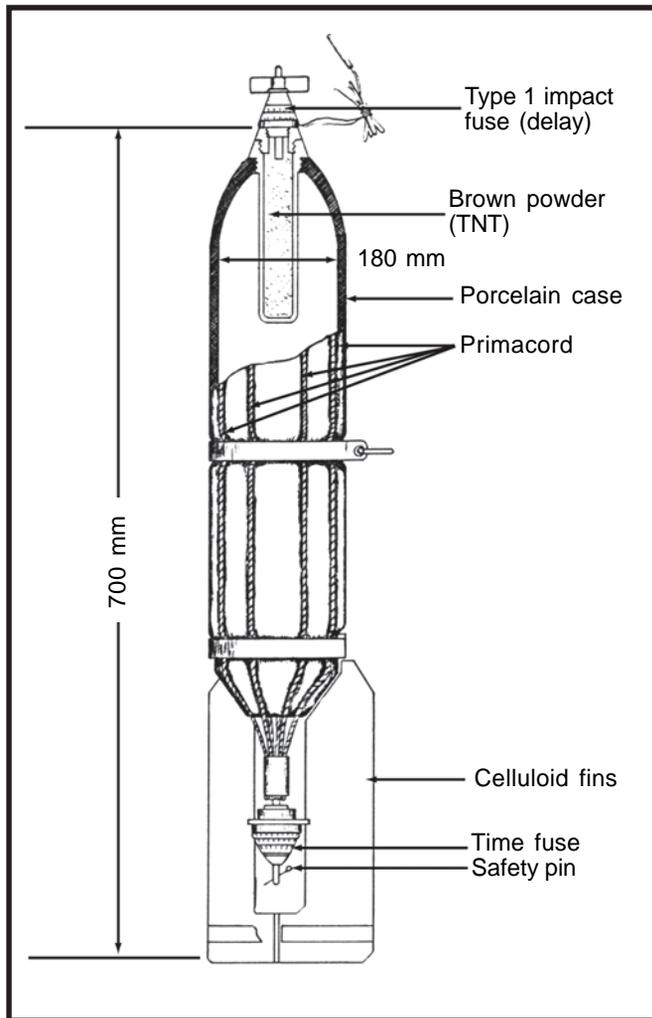
The most successful plague flea weapon developed by the Japanese was the Uji bomb. The older-type Uji, invented in 1938, was a frangible weapon with a porcelain casing and an inlaid strip of Primacord® to activate an in-



A US Army medical doctor examines a Japanese prisoner of war for signs of biological warfare vaccinations.

flight rupture and plague flea release. It weighed 55 pounds and had the capacity to hold 4.7 gallons. Early field trials demonstrated that thin-walled, metal-cased bombs required excessive quantities of explosives and thus destroyed most of the plague fleas. The Type 50 Uji bomb (introduced in 1940) contained a contact fuse that destroyed the weapon (and its contents) if it failed to burst in the air. It also weighed 55 pounds, but could hold 3 gallons. The Type 100 Uji bomb was a larger version of the Type 50, weighing 110 pounds and holding nearly 7 gallons. The Japanese considered the Type 100 inferior to the Type 50 due to its size and the possibility of damage during ordnance handling.⁷

The Type 50 Uji bomb contained about 30,000 plague fleas. Intended to burst at an altitude of 660 to 980 feet above ground level, field trials at Anta, Manchuria, concluded that 80 percent of the fleas survived dissemination and that coverage was best under conditions with high wind. The Japanese did not give up on producing



The Type 50 Uji bomb weighed 55 pounds and held 3 gallons.

the biological weapon and conducted around 4,000 dispersal trials and 2,000 human trials to demonstrate the effectiveness of the weapon.⁸

In May 1944, Unit 731 was prepared to use plague fleas against the United States in the Pacific. With the fall of their garrison at Saipan in June of that year, Unit 731 assembled a team of Soldiers to contaminate the Saipan airfield with plague fleas. A shipload of specialists and biological weapons were en route to a staging area when a US submarine sunk the ship, killing all but one crew member.⁹

In 1944, the Japanese built four gigantic submarines (the I-400 Class) that were capable of launching aircraft to bomb targets on the US West Coast and New York City. The mission, *Operation PX*, was designed to use submarines to launch biological strikes against the continental United States and the Pacific Islands.^{10, 11} In March of 1945, the Chief of Staff for the Imperial Japanese Army cancelled the mission and declared it ethically unacceptable.

In many ways, Canada was the pioneer in biological warfare for the Allies during World War II. While Great Britain and the United States only pondered the possibility of developing a weapon with psittacosis, Canada was intent on developing one. After Great Britain and the United States formally established their biological warfare programs, the Canadians worked in areas that the other two nations tended to ignore. Developing biological weapons for the vector effect was one of those areas.

While workers at Canada's Grosse Ile—a secret germ warfare research facility—labored to produce anthrax for the Allies, G.B. Reed, a professor at the Kingston Biological Warfare Laboratory on Queen's University Campus, was seeking an entomologist to develop a different class of weaponry. The intent was to create a colony of fleas for use in combination with both plague and murine typhus. This concept of using a single vector to spread two different diseases simultaneously was an innovative approach.¹²

During the Korean War, the Sino-Soviet block alleged that the United States was employing biological weapons. Contrary to the allegations made by the Sino-Soviet block during the Korean War, interest in agent-vector combinations started after the war.^{13, 14} Operation Big Itch used uninfected fleas to determine the coverage patterns and the suitability of the tropical rat flea (*Xenopsylla cheopis*, formerly termed the oriental rat flea) in terms of survival and appetite. The field trials were conducted at Dugway Proving Ground in September of 1954. The trials used guinea pigs, placed at stations along a 660-yard circular grid, to detect the presence of fleas.

Originally intended for use as an anticrop weapon, the E-14 and E-23 munitions were converted to vector munitions for the field trials. The E-14 munition was a 13-inch-diameter, 9 3/4-inch-long cardboard container with an internal actuator that released carbon dioxide, a piston that moved to expel its contents, and a small chute for clustering the E-86 aerial bomb. The E-23 munition was a 9 3/4-inch-diameter, 18-inch-long cardboard container with an external actuator that reversed a plastic bag to expel its contents. It too included a small chute for clustering the E-77 aerial bomb. Both weapons functioned at 1,000 to 2,000 feet above ground level after release from the cluster bomb, and Operation Big Itch proved a success. Using a functioning height lower than that originally intended, the weapon proved capable of covering a battalion-sized target and disrupting operations for a 24-hour period.

An important consideration was the use of carriers. The carriers allowed the fleas access to air and moisture to keep them alive during delivery. The Japanese filled their Uji bombs with sand and plague fleas. The United States considered two methods: sponge fragments and small paperboard tubes with crepe paper streams to keep the open end closed when rolled. Using the sponge fragments, the E-14 carried 100,000 fleas and the E-23 carried 200,000. Because half of the E-23s failed to function in preliminary tests, only the E-14 was used for the remainder of Operation Big Itch. The E-14 was capable of carrying 80 loop tubes, each containing 3,000 fleas.¹⁵

In the United States, the plague flea concept was competing against the use of mosquitoes, flies, ticks, and lice. Of these concepts, the United States put most of its energies behind weaponizing yellow fever in combination with the *Aedes aegypti* mosquito. The United States Navy

made significant contributions to the research in the aerosol dissemination of plague, and the British conducted a series of aerosol field trials in the Bahamas. The flea no longer attracted interest.

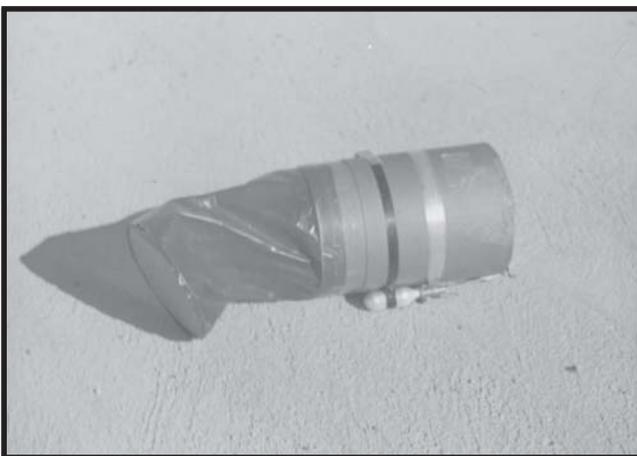
Discussion

The nuclear burst over Hiroshima on 6 August 1945 resulted in 0.13 pounds of weapon per prompt casualty. One of Japan's plague flea weapons approximated this same rate of destructiveness. With vector weapons, the issue is not the weight but the volume. For each vector, there is considerable dead volume needed to sustain life in the atmosphere.

The Japanese Uji Type 50 held 11,000 fleas per gallon of space. The United States improved the 1.5 ratio to 3.5. The E-14 could deliver fleas with a 1 percent loss in viable content, which was a significant improvement over Japan's 20 percent loss more than a decade earlier. The difference between the efforts was likely the choice of plague vectors—the human flea (used by Japan) and the tropical rat flea (considered by the United States)—and their abilities to adapt to host habitats. But regardless of the habitat, both fleas attacked people with equal vigor.

The qualities taken into consideration for biological vector weapons were the¹⁶—

- **Dispersal and flight ranges.** The maximum distance fleas tend to migrate is around 220 yards. A flea jumps up to a foot at a time and can jump more than 600 times an hour when questing for a host. In comparison, mosquitoes tend to migrate up to 1.5 miles.
- **Extrinsic-incubation periods.** The incubation period is usually temperature-dependent, but irradiation or the addition of certain chemicals can hasten the results. In ideal temperatures, a flea can transmit disease about 15 days after feeding from a plague-infected rodent.
- **Infective periods.** Fleas remain infective throughout their lifespan, but they do not transmit the disease to subsequent generations. The limiting factor is the survival rate of a blocked flea.
- **Infective threshold.** To transmit plague, a rodent must have more than 100 million organisms per milliliter of blood to be able to infect a flea during feeding.
- **Transmission rates.** Only 58 percent of tropical rat fleas are capable of spreading disease after feeding on infected animals. Other species of fleas have lower transmission rates.



The E-23 munition proved to be unreliable during Operation Big Itch.

- **Vectoral capacity.** In a blocked flea, a single bite inoculates a person with a sufficient number of microbes to result in plague infection.

In 1959, the US Army Chemical Corps board provided guidance for what has often been termed *Entomological Warfare*.¹⁷ The report acknowledged that no formal requirement existed for such weapons, but that there was a belief that guidance was necessary for research and development activities for weapon systems. Like many World War II commanders, the Chemical Corps board believed that the adoption of any vector weapon system was dependent on the persistence, predictability, and control measures.

Along with recommendations for planning aids, logistics, and employment, the Chemical Corps board considered the combinations designed specifically to confuse enemy medical and intelligence personnel (such as using a current system to deliver an incapacitating agent in place of a lethal agent, using multiple agents in a single vector type, or using a single agent with multiple vectors). The range that a vector can spread is significantly larger than what appears in nature or within the experience of medical entomology. For example, fleas can spread tularemia.¹⁸

The casualty potential of the vector effect results from calculations with finite sets. With such a small number of fleas, the expenditure is dependent on the population density of the target. The table below illustrates the hypothetical coverage properties for a vector munition (comparable to four E-14s). For comparison between flying and crawling disease vectors, the table includes a virus-mosquito combination. The information shows a hypothetical coverage of 50 percent caused by a single vector munition.

The hypothetical estimates represent a battalion-sized target, but may require layering munitions in areas with high population densities. The persistence is the length of time that the vector effect will continue to inflict casualties

before replenishment is required to maintain a barrier. Without the use of some clearance mechanism (pesticide), the target would not be safe for friendly occupation for about a week (with fleas) or a month (with mosquitoes). The minimum safe distances would be in the order of miles (due to the uncertainties of vector migration).

Conclusions

The vector effect offers biological warfare with extended options not available with the more traditional aerosol weaponry—diversifying the arsenal with additional agents and employment methods, circumventing respiratory protective means, and offering persistence to deny the utility of terrain and facilities. However, in the end, agent-vector combinations are labor-intensive, prove to be unreliable with the uncertainties of complex animal behavior, and infect limited areas (in comparison to other aerosol weapon options).

Many assume biological warfare is limited to strategic applications. The vector effect allows biological warfare to transcend through the operational phase and into tactical situations. The drawback, like any biological weapon, is the community health consequences that may persist after a conflict is resolved.

The vector effect has had its place in augmenting other weapons in a comprehensive biological arsenal (as with the United States) or as a stopgap measure when there is insufficient technology for an aerosol effect (as with Japan). Nonetheless, on its own, it represents a minor curiosity with imaginative possibilities that time and ability have passed by. ●●●

Agent-Vector Combination	Persistence (days)	Carrier					
		Sponges		Loop Tubes		Aircomb Waffles	
		Area	Density	Area	Density	Area	Density
Plague flea	1	90	35	40	180	NA	NA
Virus mosquito	15	NA	NA	40	290	90	110

Area = hectares covered
Density = maximum target of people per hectare in the target area

Endnotes:

¹Brendan Lehane, *The Complete Flea: A Light-Hearted Chronicle, Personal and Historical, of One of Man's Oldest Enemies*, Murry Publishers, 1969.

²Thomas G. Hull, *Diseases Transmitted from Animals to Man*, Charles C. Thomas Publishers, Springfield, Illinois, 1930.

³Thomas Butler, *Plague and Other Yersinia Infections, Current Topics in Infectious Disease*, Plenum Press, New York, New York, 1983.

⁴Dr. Alexandre Yersin settled in Nha Trang, Vietnam, after leaving France and established the Pasteur Institute. Dr. Yersin remains a celebrated local hero in his adopted country well after his demise. Thomas Butler's book, *ibid*, describes what the United States experienced during the Vietnam War.

⁵Vincent J. Derbes, "De Mussis and the Great Plague of 1348: A Forgotten Episode of Bacteriological Warfare" *Journal of the American Medical Association*, Vol. 19, No. 1, 4 April 1966, p. 180.

⁶Peter Williams, David Wallace, *Unit 731: Japan's Secret Biological Warfare in World War II*, The Free Press, New York, New York, 1989.

⁷Lieutenant Colonel Arvo T. Thompson, "Report on Japanese Biological Warfare (BW) Activities" Army Service Forces, Camp Detrick, Frederick, Maryland, 31 May 1946. This document contains descriptions of the biological weapons Japan investigated, along with a technical drawing based on interviews with Lieutenant General Shiro Ishii. The report is devoid of descriptions of human experiments and the use of plague fleas (revealed in US investigations in 1946 and 1947).

⁸Sheldon Harris, *Factories of Death: Japanese Biological Warfare 1932-45 and the American Cover-Up*, Routledge, New York, New York, 1994.

⁹Peter Williams, David Wallace, *Unit 731: Japan's Secret Biological Warfare in World War II*, The Free Press, New York, New York, 1989, p. 81.

¹⁰"The Transpacific Voyage of H.I.J.M.S. I-400, Tom Paine's Journal: July 1945 to January 1946," 1991, <<http://www.pacerfarm.org/i-400/>>, accessed on 1 August 2005.

¹¹Hawaiian Undersea Research Laboratory, "I-401 Submarine Found Off of Barbers Point," 2005, <<http://www.soest.hawaii.edu/HURL/I-401.html>>, accessed on 1 August 2005.

¹²John Bryden, *Deadly Allies: Canada's Secret War 1937-1947*, McClelland & Stewart Inc., Toronto, Canada, 1989, p. 111.

¹³The Sino-Soviet allegations remain the stated historical policy of North Korea and China, but they have been rather explained away by most scholars. Central to the allegations is the fact that the United States granted Japanese Biological Warfare researchers immunity from war crimes prosecution at the end of the World War II.

¹⁴Kathryn Weathersby and Milton Leitenberg, "New Evidence on the Korean War," *Cold War International History Project Bulletin 11*, George Washington University, 1999, pp. 176-199.

¹⁵Biological Warfare Assessment Laboratories, "Suitability Test of E-14 and E-23 Munitions as Disseminators of Arthropod Vectors. BW 1-55, Operation Big Itch," BWALR 6-A, US Army Dugway Proving Ground, Utah, 17 November 1954.

¹⁶William Rose, "An Evaluation of Entomological Warfare as a Potential Danger to the United States and European NATO Nations," DPG-S-445A, US Army Dugway Proving Ground, Utah, March 1981, pp. 33-34.

¹⁷Chemical Corps Board, "Insect-Borne Antipersonnel BW," Army Chemical Center, Maryland, 1 May 1959.

¹⁸Frank Prince, "Tularemia, Attempted Transmission by Each of Two Species of Fleas: *Xenopsylla cheopis* (Rotts) and *Diamanus montanus* (Baker)," Government Printing Office, Washington, D.C., 1946.

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Repair of the Chemical- Agent Monitor Simulator (CAMSIM)



Ahtna Development Corporation, through the Program Executive Office for Simulation, Training, and Instrumentation (PEO STRI) in Orlando, Florida, has been awarded the contract for maintenance of the CAMSIM. For additional information on CAMSIM repair, contact Ron Richards at e-mail <Ronald.Richards@peostri.army.mil> or Milton Cates at <Milton.Cates@peostri.army.mil>, or call (407) 384-3613/3717.